# **Upper Extremity Motion Assessment in Adult Ischemic Stroke Patients: A 3-D Kinematic Model**

J. Van Bogart<sup>1</sup>, J. McGuire<sup>2</sup>, G. F. Harris<sup>1,3</sup>

<sup>1</sup>Department of Orthopaedic Surgery, Medical College of Wisconsin, WI, USA
<sup>2</sup>Department of Physical Medicine and Rehabilitation, Medical College of Wisconsin, WI, USA
<sup>3</sup>Department of Biomedical Engineering, Marquette University, WI, USA

Abstract – As part of a larger evaluative study of the effects of botulinum toxin type A (BTA) in ischemic stroke patients, a kinematic model of the trunk and upper extremities (UE) has been developed. The 3-D model provides a comprehensive method of assessing UE motion during performance tasks including exercises in reaching, grasping, and releasing. The 17-marker system tracks UE motion at a rate of 120 SPS with 7 infrared CCD cameras. The biomechanical model developed for the system allows expression of torso, shoulder, elbow, and wrist motion in terms of Euler expressions. Concurrent EMG data is used to confirm periods of co-contraction and spasticity during planned movement. Preliminary trials with the system indicate sufficient fidelity for continued clinical trials.

Keywords - Botox, motion analysis, hemiplegia, stroke

## I. INTRODUCTION

Recovery from ischemic stroke has been explained by patients learning new skills, by ischemically injured cells recovering, but more recently, by brain plasticity. The latter is now the subject of interest and offers a potential for new and innovative treatment strategies. Neuro-plasticity means that a re-ordering of neuronal patterns can be affected by internal and external factors, including training. Whether the development of spasticity itself negatively affects neural re-organization or whether it is an effect of neural reorganization is an important theoretical question but also a question important for management decisions. Today there are powerful anti-spasticity measures, such as botulinum toxin type A intra-muscular blockade (BTA), which have become the standard of care for spasticity management [1, 2, 3, 4, 5, 6]. These powerful measures not only provide new management strategies, but may also help solve unanswered questions.

This study begins to quantitatively measure the central and peripheral effects of clinically administered BTA in a small sample of ischemic stroke patients both before and after injection. The hypothesis is entered that central reorganization of injured neural tissue and connections is negatively affected by immobility including spasticity, which further limits mobility and inhibits motor recovery. Furthermore, it is proposed that early spasticity management can enhance motor recovery in the upper extremity after ischemic stroke with intramuscular botulinum to a degree that can be detected quantitatively clinically, functionally, biomechanically and from direct brain measurements by f-MRI. The corollary is that aggressive spasticity reduction by BTA combined with mobilization could positively influence peripheral function as well as central motor activity.

This paper addresses the quantitative assessment of upper extremity kinematics in the ischemic stroke patients before and after treatment with BTA.

## II. METHODOLOGY

## A. Biomechanical Testing

A biomechanical model of the trunk and upper extremities has been developed to perform a comprehensive 3-D kinematic analysis. Biomechanical testing includes measuring passive resistance through range of motion at the elbow using a Biodex Multi-Joint System 3 (Biodex Medical Systems, Shirley, NY). Kinematics are analyzed during a short series of upper extremity performance tasks that include exercises in reaching, grasping, and releasing. Subjects are asked to: 1) move objects from one quadrant to another on a tabletop, 2) to reach and grasp a book from a shelf at eye level and 3) perform rapid repetitive elbow extensions. Kinematic analysis of these tasks includes threedimensional analysis of trunk and upper extremity displacement as well as velocity and acceleration. kinematic testing is performed while seated and under constant supervision of laboratory personnel.

Concurrently, muscle activity is recorded during all biomechanical testing using a ten-channel surface EMG system (Motion Lab Systems, Inc., Baton Rouge, LA). Surface electrodes are attached to predetermined sites using hypoallergenic tape. Sites chosen for EMG analysis include: teres major, pectoralis major, deltoid, medial and lateral heads of the triceps, biceps brachii, brachioradialis, pronator teres, wrist extensors and wrist flexors.

Kinematic data of the upper extremities is obtained with a Vicon 524 (Oxford Metrics, Oxford, England) 3-D motion analysis system using seven infrared CCD cameras and retro-reflective markers. Each camera in the system collects kinematic marker data at 120Hz. With this system, lightweight reflective markers are attached to the skin at predetermined anatomic landmarks (over the olecranon process for example) using double-sided hypoallergenic tape. While the subject is performing the selected activities in the field of view of the cameras, the system records movement of the limb segments by "tracking" the three-dimensional (3-D) location of the markers. Prior to being

Report Documentation Page			
Report Date 25OCT2001	Report Type N/A	Dates Covered (from to)	
Title and Subtitle Upper Extremity Motion Assessment in Adult Ischemic Stroke Patients: A 3-D Kinematic Model		Contract Number	
		Grant Number	
		Program Element Number	
Author(s)		Project Number	
		Task Number	
		Work Unit Number	
Performing Organization Name(s) and Address(es) Department of Orthopaedic Surgery, Medical College of Wisconsin, WI,		Performing Organization Report Number	
Sponsoring/Monitoring Agency Name(s) and Address(es) US Army Research, Development & Standardization Group (UK) PSC 802 Box 15 FPO AE 09499-1500		Sponsor/Monitor's Acronym(s)	
		Sponsor/Monitor's Report Number(s)	
<b>Distribution/Availability Sta</b> Approved for public release, d			
_		EEE Engineering in Medicine and Biology Society, 001351 for entire conference on cd-rom.	
Abstract			
<b>Subject Terms</b>			
Report Classification unclassified		Classification of this page unclassified	
Classification of Abstract unclassified		Limitation of Abstract UU	
Number of Pages 3			

processed by the kinematic model, all marker coordinate data are low-pass filtered using a 6Hz, dual-pass, 2<sup>nd</sup> order Butterworth filter.

Marker #	Anatomic Location
1,7	Acromion process
2,8	Mid-humeral shaft
3,9	Lateral humeral condyle
4,10	Ulnar styloid
5,11	Radial styloid
6,12	3 <sup>rd</sup> Metacarpal
13,14	Mid-Clavicle shaft
15	Sternal notch
16	Lower sternum
17	Spinous process of 7 <sup>th</sup> cervical vertebrae (not shown)

Table 1 Description of marker locations.

## B. Kinematic Model

The kinematic model is established using the laboratory referenced 3-D marker locations provided by the VICON 524 system. Table 1 and figure 1 describe the anatomic marker locations used by the model. These marker locations segment the upper body into seven distinct segments a torso, right and left hands, forearms, and upper arms.

The 3-D coordinates were used to create orthogonal body coordinate systems, which were directed from the distal to the proximal segment. Typically, one unit vector was defined along the segment long axis. A second unit vector was then defined perpendicular to the first, passing through the joint center. The third and final unit vector was then constructed perpendicular to the first two using vector cross products.

The model outputs include Euler angle orientations of each segment relative to its next proximal segment. That is, the hand motion is relative to the forearm motion, the forearm relative to the humerus and so on. The torso's orientation, however, is relative to the laboratory's global coordinate system. Relative orientations are described for each of the anatomic planes: sagittal, coronal and transverse.

# III. RESULTS

Model application is depicted in Figure 2, which shows the flexion extension kinematics of a subject's hemiplegic side both prior to the BTA injection and approximately 30 days following the injection. The exercise depicted in this graph is the subject performing five successive elbow flexion/extension cycles with the shoulder abducted at approximately 90 degrees. Positive slopes in the graph denote flexion while negative slopes denote extension; with peaks occurring when there is a direction change. Figure 3 shows surface EMG traces associated with the kinematics found in figure 2.

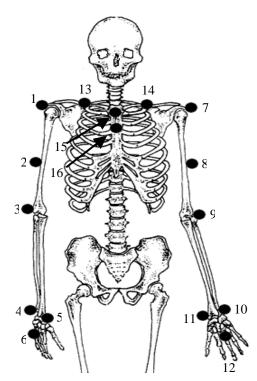


Figure 1 Marker locations for upper body kinematic model.

## IV. DISCUSSION

Interpreting these traces reveals an increase in total range of elbow flexion/extension motion following the BTA injection. This is evidenced by larger "peak to valley" excursions in the post-injection trace. The apparent offset between the two curves is due to a more relaxed starting posture following the treatment. This patient population is prone to a flexion posture in the upper extremity when untreated, hence prior to treatment the average elbow flexion/extension angle is offset by approximately 20 degrees less than after treatment.

The post-injection EMG graph shows a more distinguished cyclic firing pattern in the medial triceps of the hemiplegic arm during this exercise. This seems to indicate a slower more deliberate movement. This increased control is supported by figures 2 and 3 as well as video tape analysis.

#### V. CONCLUSION

Based on preliminary data, this methodology shows promise for quantifying motion aspects of the motor effects of BTA in hemiplegic stroke patients. At this time, kinematic and electromyographic analyses have been done in a pilot sample. On the basis of these results, the biomechanical (kinematic) system is appropriate for further clinical implementation.

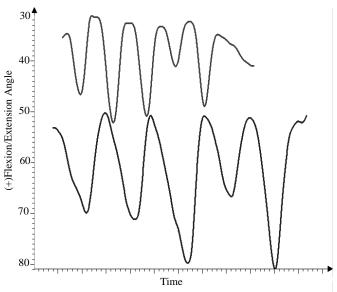
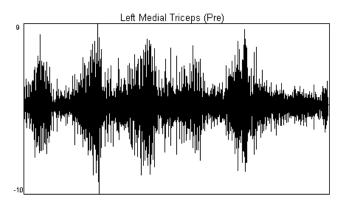


Figure 2 Pre (top trace) and post (bottom) BTA injection elbow kinematics.



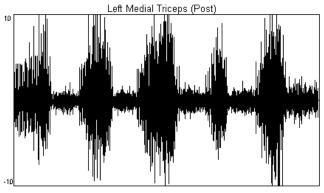


Figure 3 Pre and post BTA injection medial triceps EMG recordings.

#### ACKNOWLEDGMENT

Special thanks to the subjects willing to participate in our study, Kelly Myers for her much needed assistance, the Orthopaedic Rehabilitation and Engineering Center (OREC) at Marquette University and the Medical College of Wisconsin and to Allergan, Inc.(Irvine, CA), makers of BOTOX®, for their sponsorship.

#### REFERENCES

- [1] Gracies JM, Nance P, Elovic E, McGuire J, Simpson DM, "Traditional pharmacological treatments for spasticity Part I: Local treatments. Spasticity: Etiology, Evaluation, Management, and the Role of Botulinum Toxin Type A," *Muscle and Nerve*, Supp 6, 1997, S61-S91.
- [2] Simpson DM, "Clinical trials of botulinum toxin in the treatment of spasticity. Spasticity: Etiology, Evaluation, Management, and the Role of Botulinum Toxin Type A," *Muscle and Nerve*, Supp 6, 1997, S619-S75.
- [3] Simpson DM, "Patient visit forms and rating scales: Spasticity: Etiology, Evaluation, Management, and the Role of Botulinum Toxin Type A," *Muscle and Nerve*, Supp 6, 1997, Appendix.
- [4] Pierson SH, "Outcome measures in spasticity measurement. Spasticity: Etiology, Evaluation, Management, and the Role of Botulinum Toxin Type A," *Muscle and Nerve*, Supp 6, 1997, S36-S60.
- [5] Pierson SH, Katz DI, Tarsy D, "Botulinum toxin A in the treatment of spasticity: functional implications and patient selection," *Arch Phys Med Rehabil*, 77, 1996: 717-721.
- [6] Simpson DM, "Clinical trials of botulinum toxin in the treatment of spasticity," *Muscle and Nerve*, 20 (suppl), 1997: S169-S175.
- [7] Lee KC, Carson L, Kinnin E, Patterson V, "The Ashworth scale: a reliable and reproducible method of measuring spasticity," *J Neuro Rehab*, 3, 1989: 205-209.